Household Model of Chagas Disease Vectors (Hemiptera: Reduviidae) Considering Domestic, Peridomestic, and Sylvatic Vector Populations

L. STEVENS,[1,2] D. M. RIZZO,[3] D. E. LUCERO,[1] AND J. C. PIZARRO[4]

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ABSTRACT Disease transmission is difficult to model because most vectors and hosts have different generation times. Chagas disease is such a situation, where insect vectors have 1–2 generations annually and mammalian hosts, including humans, can live for decades. The hemataphagous triatominae vectors (Hemiptera: Reduviidae) of the causative parasite *Trypanosoma cruzi* (Kinetoplastida: Trypanosomatidae) usually feed on sleeping hosts, making vector infestation of houses, peridomestic areas, and wild animal burrows a central factor in transmission. Because of difficulties with different generation times, we developed a model considering the dwelling as the unit of infection, changing the dynamics from an indirect to a direct transmission model. In some regions, vectors only infest houses; in others, they infest corrals; and in some regions, they also infest wild animal burrows. We examined the effect of sylvatic and peridomestic vector populations on household infestation rates. Both sylvatic and peridomestic vectors increase house infestation rates, sylvatic much more than peridomestic, as measured by the reproductive number R_0. The efficacy of manipulating parameters in the model to control vector populations was examined. When $R_0 > 1$, the number of infested houses increases. The presence of sylvatic vectors increases R_0 by at least an order of magnitude. When there are no sylvatic vectors, spraying rate is the most influential parameter. Spraying rate is relatively unimportant when there are sylvatic vectors; in this case, community size, especially the ratio of houses to sylvatic burrows, is most important. The application of this modeling approach to other parasites and enhancements of the model are discussed.

KEY WORDS triatoma, Chagas disease, mathematical model, vector control

Chagas disease or American trypanosomiasis is caused by the parasite *Trypanosoma cruzi* (Kinetoplastida: Trypanosomatidae) and afflicts up to 10 million people in Latin America (Hotez et al. 2008). It causes potentially life-threatening disease in up to one-third of those infected and is most easily cured during the early stage of infection by using drugs with considerable side effects (Andrade et al. 2011). The most effective way to achieve and maintain low prevalence of Chagas disease is to prevent humans from being bitten by infected insect vectors. There are >130 species in the subfamily Triatominae (Hemiptera: Reduviidae), idiomatically referred to as vinchucas, chinches, and kissing bugs in South, Central, and North America, respectively (Capinera 2008). Interrupting transmission is complicated by the fact that the parasite has multiple mammalian hosts and insect vectors. Triatominae feed on a variety of vertebrate hosts, including mammals and birds, yet only mammals can become infected. Because vectors feed on a variety of hosts, they have domestic, peridomestic (around the house), and sylvatic (wild) populations. The ecology of the parasite life cycle varies among regions, being influenced by the local domestic vertebrates, insect vector species, and zoonotic vertebrate hosts. In some regions, sylvatic vector populations likely jeopardize control efforts (Noireau 2009). Research to date suggests low-cost economic and human behavior strategies, in combination with insecticide spraying and housing improvement, are the most effective way to reduce disease transmission (Bustamante et al. 2009, Monroy et al. 2011, Lucero et al. 2013).

Infection in the vertebrate host occurs when the parasite enters the body through mucous membranes, a break in the skin, or the ingestion of infected vectors. The blood-sucking vectors live in houses, moving from diurnal hiding places to feed at night. Scratching the itching insect bite transmits the parasite into the blood of the mammalian host. The parasite can also be transmitted to humans by blood transfusion, and maternal–fetal transmission occurs. Severe acute symptoms occur in ≈1% of cases. Chronic symptoms develop in about one-third of those infected and occur some 10–20 yr after initial infection (Andrade et al. 2011). The disease affects the heart in 25% of those infected, the esophagus and colon in 6%, and the peripheral nervous system in 3%.

Three initiatives have been developed to reduce the prevalence of Chagas disease in Latin America. The

[1] Department of Biology, University of Vermont, 321 Marsh Life Science Building, Burlington, VT 05405.
[2] Corresponding author, e-mail: lori.stevens@uvm.edu.
[3] Facultad de Bioquímica, Universidad Mayor Real y Pontificia de San Francisco Xavier de Chuquisaca, Sucre, Bolivia.
[4] School of Engineering, University of Vermont, 220 Votey Building, Burlington, VT 05405.

Southern Cone Initiative was implemented in 1991 in Argentina, Bolivia, Brazil, Chile, Paraguay, and Uruguay to reduce Chagas disease by targeting transmission by insect vectors and blood transfusion. Five of the six countries reduced prevalence by 60–90%, so that rates of infection in 2000 ranged from a low of 0.06% in Uruguay to a high of 3.9% in Paraguay (Moncayo and Silveira 2009). Despite this success, some regions, especially the Department of Chuquisaca, Bolivia, retain a high rate of infection. In regions where the *Triatoma infestans* (Reduviidae, Triatominae) is solely domestic, vector control is simplified. Genetic evidence suggests the Andes region is possibly the geographic origin of the species (Giordano et al. 2005); however, the Gran Chaco has not been ruled out (Perez de Rosas et al. 2011, Piccinali et al. 2011, Quisberth et al. 2011). Within Chuquisaca, in the Province of Oropeza, 35% of children aged <5 yr were seropositive in 2002 (SEDES-Chiquisaca 2006). *T. infestans*, the major vector of Chagas disease in the Southern Cone countries, has been increasingly reported from sylvatic habitats (Buitrago et al. 2010; Waleckx et al. 2011, 2012). Since 1997, the Andean (Colombia, Ecuador, Peru, and Venezuela) and Central American (Belize, Costa Rica, El Salvador, Guatemala, Honduras, Mexico, Nicaragua, and Panama) initiatives have focused on vector control in these regions where there are domestic, peridomestic, and sylvatic populations.

Although vectors feed on a variety of hosts and potentially have domestic, peridomestic, and sylvatic populations, most triatomine species are strictly inhabitants of wild ecotopes and never invade houses. A small number are domesticated, and along with a number of sylvatic species that intermittently invade houses, these transmit the parasite to humans and domestic mammals. Vector control is central to reducing transmission because home infestation rates can be high and 100s of triatomines can be found in a single house (Pizarro et al. 1996). The main domestic species (*T. infestans*, *Triatoma brasiliensis*, *Triatoma dimidiata*, *Triatoma sordida*, *Panstrongylus megistus*, and *Rhodnius prolixus*) are responsible for >80% of human Chagas disease (Stevens et al. 2011).

Chagas disease has been modeled as a vector-transmitted disease by using indirect transmission models (Anderson and May 1991) by several authors (Velasco–Hernandez 1994, Cohen and Gurtler 2001, Inaba and Sekine 2004, Spagnuolo et al. 2011, Cruz–Pacheco et al. 2012). These models consider the transmission dynamics between the mammalian host and the insect vectors. The more recent models have focused on the effect of various control measures, including the effect of insecticide spraying on vector control (Spagnuolo et al. 2011, Cruz–Pacheco et al. 2012), and considered transmission dynamics among various populations such as humans, domestic animals (e.g., dogs), peridomestic animals (e.g., chickens), and vectors. One of the main difficulties when modeling vector-transmitted diseases is that the time scales of the host and vector population cycles are very different. Human life expectancy is ≈70 yr in many of these countries, but vector life cycles are typically of the order of months.

Time-related complications could be avoided by using a different modeling approach, that is, by considering house infestation as the phenomenon to model. In the following model, we consider the dwellings of the mammals that suffer the disease to be the "hosts," that is, hosts include human houses, peridomestic structures (e.g., corrals), and sylvatic dwellings (e.g., animal nests, caves, and rodent burrows). This approach is similar to that used to model transmission of bovine viral diarrhea among farm animals (Tinsley et al. 2012).

Recently developed molecular genetic techniques can be used to estimate vector movement between habitats. Data using microsatellite loci provide information on the geographic scale at which insects randomly mate and can be used to estimate migration (Dorn et al. 2003, Calderon et al. 2004, Pizarro et al. 2008). In addition, methods are available to identify the sources of vertebrate bloodmeals (Pizarro et al. 2007, Pizarro and Stevens 2008, Stevens et al. 2012). By using these techniques, vector movement can be estimated, for example, from the number of domestic vectors that contain blood from peridomestic areas (Pizarro and Stevens 2008). We developed a model that includes vector movement between domestic and peridomestic or sylvatic habitats, a major concern of Chagas disease transmission. Our model (Fig. 1) is based on the observation that among rural communities in the Province of Zudañez, Department of Chiquisaca, Bolivia, data from 2002 show a significant correlation between the percent of houses infested and Chagas disease prevalence in children aged <5 yr ($r^2 = 0.88$; $P < 0.02$), whereas no correlation exists between the percent of infected triatomines and disease prevalence ($P > 0.50$) (SEDES-Chiquisaca 2006). With this model, we ask: what is the effect of peridomestic or sylvatic vector populations on the number of infested houses? In addition, we perform a sensitivity analysis (i.e., examination of how the dynamics of the system change as parameter values vary) to provide information on the most effective methods for controlling vector populations.

Materials and Methods

Model Development. The direct transmission model considers susceptible (uninfested), infested, and resistant (insecticide-treated) houses. With this approach, we proceed to model transmission as it is done for macroparasitic diseases, that is, following the vector burden of each house or corral: the greater the vector burden, the higher the infestation of the dwelling. The model assumes mass action, which implies that vectors are equally mobile throughout the area, and has two groups of vectors based on the habitat where they feed and reproduce. One habitat is houses; the other comprises peridomestic structures (i.e., corrals or other structures where livestock are kept, or sylvatic dwellings such as wild animal burrows). The model includes the common control method for Chagas disease vectors, periodic spraying of residual insecticides, and there may or may not exist differences in application to the houses and peridomestic areas.

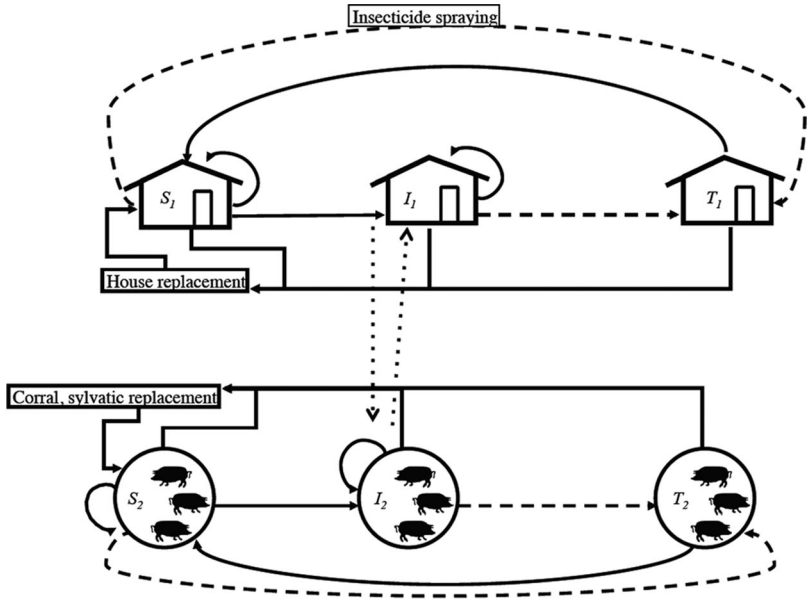

Fig. 1. Diagram of household-level direct transmission model with compartments for *S*usceptible, *I*nfested and *T*reated houses (indicated by subscript 1) and corrals or sylvatic structures (subscript 2). Arrows show possible transitions of houses between compartments, where solid lines indicate movement of vectors among the same type of structure, dashed lines indicate insecticide spraying, and dotted arrows indicate movement between houses and corrals or sylvatic structures and vice versa.

For our model, the reproduction number represents the number of structures a single infested house can infest. In the following text, we describe the model, calculate the reproduction number, and examine house infestation under a variety of conditions.

Movement between houses and peridomestic areas is modeled such that vectors residing in houses (group 1) spend, on average, $1/\tau_1$ units of time in corrals or burrows, and (group 2) likewise, vectors in corrals or burrows spend, on average, $1/\tau_2$ units of time in houses. The equations describing the interaction between house and peridomestic vectors are:

$$\frac{dS_i}{dt} = \mu_i N_i - \beta_i S_i\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right] - (\mu_i + \gamma_i)S_i + \theta_i T_i$$

$$\frac{dT_i}{dt} = \gamma_i(I_i + S_i) - \sigma_i\beta_i T_i\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right] - (\theta_i + \mu_i)T_i$$

$$\frac{dI_i}{dt} = \beta_i(S_i + \sigma_i T_i)\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right] - (\mu_i + \gamma_i)I_i$$

where for $i, j = 1, 2$ and $i \neq j$; S_i, I_i and T_i represent the number of susceptible, infested, and insecticide-treated dwellings in each group (1 = houses, 2 = corrals or sylvatic), respectively; and $N_i = S_i + T_i + I_i$ (Fig. 2). The parameters β_i and μ_i represent the dwelling infestation and replacement rates, respectively. In addition, let:

$$\eta_i = \frac{\tau_i}{\tau_i + \mu_i}$$

and

$$N_i^* = (1-\eta_i)N_i + \eta_j N_j$$

represent the fraction of their lives that vectors in group i spend in group j, and the weighted effective population size in each group when receiving nonlocal vectors ("visitors" of the other group), respectively. The vaccination or insecticide spraying-related parameters for each subgroup are γ_i, θ_i, and σ_i, which are the spraying rate, insecticide waning rate, and spraying efficacy coefficients, respectively (Fig. 2). Dwellings, whether infested or not, are sprayed periodically at the same rate (γ_i). Waning (θ_i) represents the amount of time after application that the insecticide is effective. As spraying may not always kill all the insects in a dwelling, σ_i is the proportion of dwellings that is insect-free after spraying and (1-σ_i) is the proportion of dwellings that is still infested after spraying.

We used MATLAB (version 7.10.0 R2010A, Natick, MA) to determine that if the total number of dwellings is constant, the model reduces to:

$$\frac{dT_i}{dt} = \gamma_i(I_i + S_i) - \sigma_i\beta_i T_i\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right] - (\theta_i + \mu_i)T_i$$

$$\frac{dI_i}{dt} = \beta_i(S_i + \sigma_i T_i)\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right] - (\mu_i + \gamma_i)I_i.$$

The basic reproductive number, R_0, is the average number of susceptible dwellings successfully infested

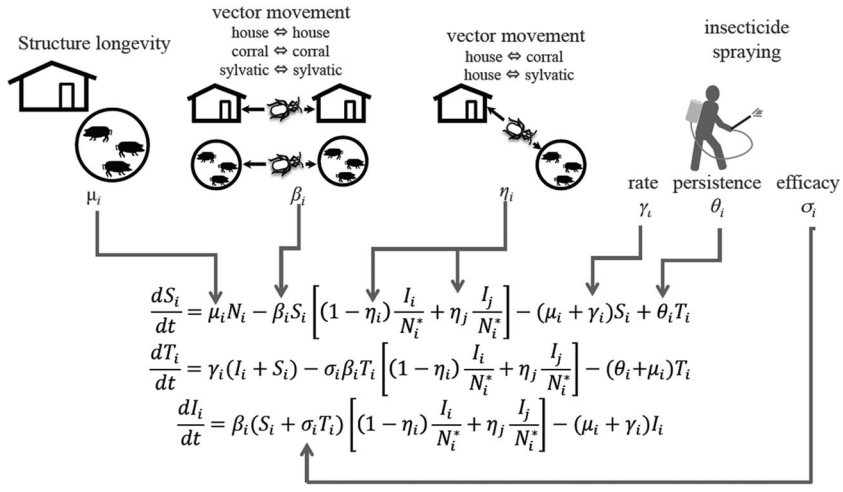

Fig. 2. Parameterization of the household transmission model determines rates of transition among compartments. Included are parameters, defined in Table 1, for *S*usceptible, *I*nfested, and *T*reated houses, as shown in Fig. 1. $N_i = S_i + T_i + I_i$, where $i = 1$ refers to the houses, two refers to corrals or sylvatic structures.

by each infested dwelling in the absence of insecticide treatments. We define the parameter R_T as the reproductive number when insecticides are applied. If $R_T > 1$, the infested houses are increasing. The goal of vector control is to find parameters such that $R_T < 1$. To compute the reproduction number, we construct a mapping, the next-generation operator, from the original equations. Assuming that at the start of the epidemic, S_i, the number of susceptible dwellings remains constant, we need only consider the dynamics of treated and infested dwellings, T_i and I_i, that actively engaged in vector infestation. Setting the two previous equations to zero and rearranging yields:

$$(\theta_i + \mu_i)T_i = \gamma_i(I_i + S_i) - \sigma_i\beta_i T_i \left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j \frac{I_j}{N_i^*}\right],$$

and

$$(\mu_i + \gamma_i)I_i = \beta_i(S_i + \sigma_i T_i)\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j \frac{I_j}{N_i^*}\right],$$

which, after further rearrangement, yields the fixed point system:

$$\Phi(x) = x$$

where

$$x = (T_1, I_1, T_2, I_2)',$$

and

$$\Phi(x) = \begin{bmatrix} \dfrac{\gamma_i(I_i + S_i) - \sigma_i\beta_i T_i\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right]}{(\theta_i + \mu_i)T_i} \\ \dfrac{\beta_i(S_i + \sigma_i T_i)\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right]}{(\mu_i + \gamma_i)I_i} \\ \dfrac{\gamma_i(I_i + S_i) - \sigma_i\beta_i T_i\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right]}{(\theta_i + \mu_i)T_i} \\ \dfrac{\beta_i(S_i + \sigma_i T_i)\left[(1-\eta_i)\frac{I_i}{N_i^*} + \eta_j\frac{I_j}{N_i^*}\right]}{(\mu_i + \gamma_i)I_i} \end{bmatrix}.$$

Table 1. Parameter values used in the simulations of the direct transmission model

Parameter	Control: no movement between houses and corrals	Case 1: vectors move between houses and corrals	Case 2: vectors move between houses and sylvatic	Definition
η_i	0/0	0.20/0.20	0.10/0.50	Fraction of their life vectors of group i spend in group j
N_i	100/100	100/100	50 of each	Total no. of dwellings
μ_i	0.05/0.05	0.05/0.05	0.05	Natural mortality rate of dwellings
β_i	1/2	1/2	0.01/0.12	Infestation rate of dwellings
γ_i	1/1	1/1	Annually	Spraying rate
θ_i	2/12	2/12	3 mo inside	Waning rate
σ_i	0.10/0.80	0.10/0.80	0.10/0	Spraying efficacy (percent infested after spraying)

Each row lists a parameter, the values used in the different model cases studied, and the parameter definition. For each case, the values in houses and corrals or sylvatic areas are shown, separated by the shilling (/) symbol.

Finding the fixed points of this map is equivalent to finding the equilibrium points of the original differential equation system. Linearizing around the infestation-free fixed point and computing the spectral radius of the linearized map, we obtain the reproduction number:

$$R_0 = \frac{1}{2}\left|\frac{(1-\eta_1)\beta_1 c_1 T_1}{a_1 b_1 N_1} + \frac{(1-\eta_2)\beta_2 c_2 T_2}{a_2 b_2 N_2} + \sqrt{\left\{\frac{4\eta_1\eta_2 T_1 T_2 \beta_1 \beta_2 c_1 c_2}{a_1 a_2 b_1 b_2 N_1 N_2} + \left[\frac{(1-\eta_1)\beta_1 c_1 T_1}{a_1 b_1 N_1} + \frac{(1-\eta_2)\beta_2 c_2 T_2}{a_2 b_2 N_2}\right]^2\right\}}\right|.$$

where $a_i = (\mu_i + \gamma_i)$, $b_i = (\mu_i + \gamma_i + \theta_i)$ and $c_i = (\mu_i + \gamma_i\sigma_i + \theta_i)$.

Model Parameterization and Sensitivity Analysis. Simulations and sensitivity analyses were performed under several assumptions and are illustrated over a time horizon of 10 yr, a reasonable timeframe to examine the initial effects of different insecticide treatment strategies and to observe approach to equilibrium dynamics.

The parameters and parameter values (Table 1) were estimated for the Department of Chuquisaca, Bolivia, through contacts with local government health officials. For example, surveys found vectors in 13% of houses after 5 yr of annual spraying (SEDES-Chiquisaca 2006). We provide the model assumptions later in the study.

Vector movement between houses and peridomestic structures is symmetrical, and ≈20% of vectors move between ecotopes. There are 100 houses per community. In the model, houses are assumed to have a 20-yr lifespan. In many parts of this region, new corrals are constructed along with new houses. In other areas, when people construct a new house, the abandoned structure is used to shelter peridomestic animals; thus, domestic and peridomestic dwellings are assumed to be equally abundant, have the same life span, and have the same infestation rates (i.e., colonizing insects do not distinguish between domestic and peridomestic structures). Sylvatic dwellings are as common as houses. Houses and peridomestic structures are sprayed annually; sylvatic dwellings are never sprayed. Residual insecticides are effective in houses for ≈6 mo; however, in peridomestic structures with more exposure to climatic conditions, we assume immunity lasts about 1 mo. We assume 90% of sprayed houses contain no insects, but spraying is assumed to kill all the insects in only 20% of peridomestic structures. After the initial simulations, we did a sensitivity analysis, examining the effects of a range of parameter values on R_T.

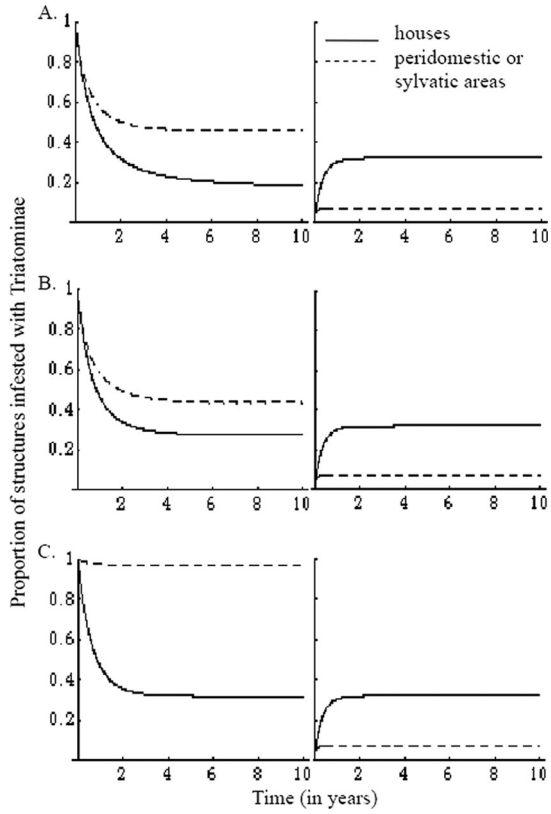

Fig. 3. Proportion of houses and peridomestic or sylvatic structures infested (left) or insecticide-treated (right) over time for three cases of the model. Initially, all structures are infested. (A) The results with no movement between houses and corrals as a basis for comparison. (B) The results where vectors move between houses and corrals. (C) The results when vectors move between houses and sylvatic. The simulations show that the proportion infested changes as a function of vector movement and insecticide spraying rates (sylvatic environments are not sprayed); however, the proportion treated is not affected. Parameter values are in Table 1.

Results

The main conclusion is that insecticide persistence and spraying frequency have the largest influence on house infestation. In the following text, we elaborate on the evidence to support this claim.

The percentages of infested houses were examined for three cases: the presence of peridomestic structures, the occurrence of sylvatic populations, and the effect of different spraying programs. First, we determined the dynamics of infestation by using the direct transmission model with no movement between houses and corrals. We then compared this result with the case with symmetrical migration between the houses and peridomestic areas and examined the effect of sylvatic dwellings that are never sprayed. In addition to examining house infestation rates, we examined the reproductive rate R_0.

We modeled infestation by using the direct transmission model with no movement between houses and

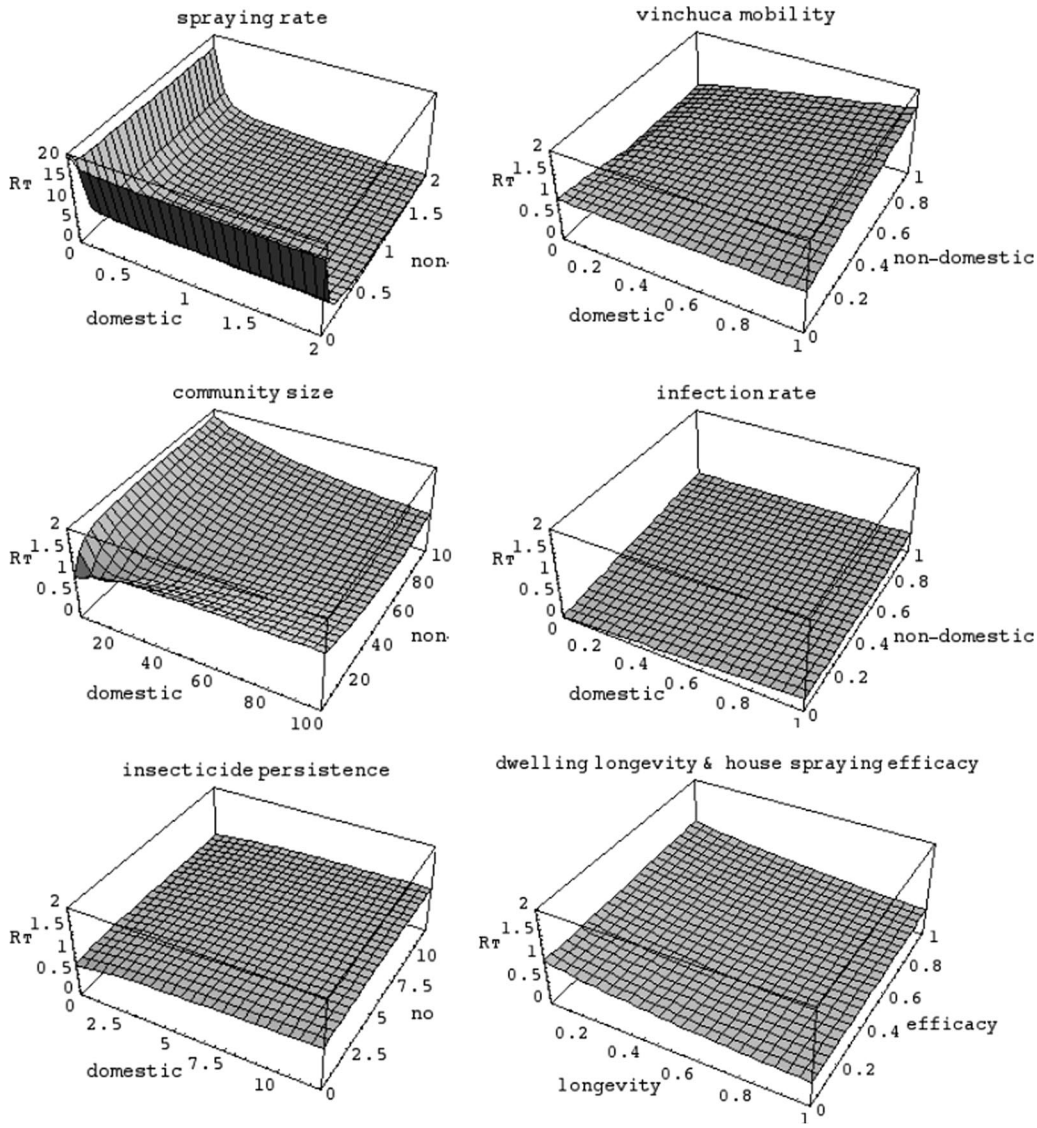

Fig. 4. R_0 over a range of parameters for the case of houses and peridomestic structures.

corrals as the basis for comparison (Fig. 3A). The simulation results show that insecticide treatment does indeed reduce infestation; however, even after 10 yr, 19% of houses and 46% of peridomestic structures are infested. When we add migration between the houses and peridomestic areas (Fig. 3B), house infestation increases from 19 to 25%, whereas peridomestic infestation decreases from 46 to 41%. Next we examine the effect of sylvatic areas that are never sprayed (Fig. 3C); this results in the highest infestation rates, 33%, for houses, and almost all burrows are infested.

In addition to examining house infestation rates, we examined the reproductive rate R_0. First we consider domestic and peridomestic areas that differ in the insecticide persistence time and spraying efficacy (Fig. 4). For all parameters, except spraying rate, R_0 (z-axis) varies from 0 to 2; however, R_0 is almost 10-fold higher when dwellings are sprayed infrequently or not at all (z-axis ≈20 for spraying rate). Overall we see that R_0 is most sensitive to spraying rate, shows intermediate sensitivity to community size (number of houses) and vector mobility, and is least affected by infestation rate, insecticide persistence, dwelling longevity, and spraying efficacy.

In the case where there are sylvatic vectors (Fig. 5), the reproductive rate R_0 is about an order of magnitude higher than when there are no sylvatic vectors and shows little sensitivity to house spraying rate. It is most sensitive to dwelling longevity and the size and mobility of the sylvatic vector population. As with the peridomestic case, there is little effect of insecticide persistence and spraying efficacy, whereas infestation rate has an increased effect.

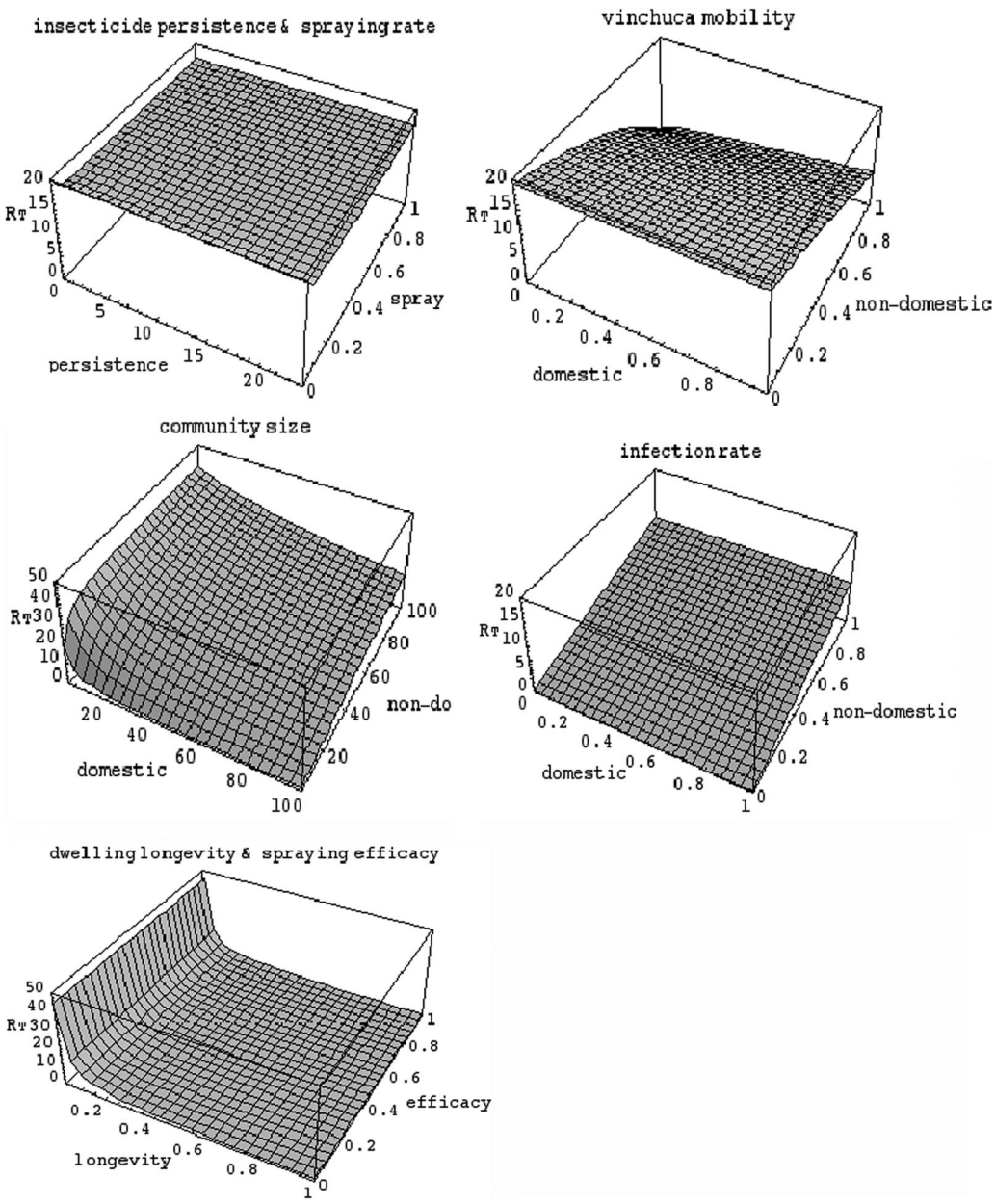

Fig. 5. R_0 over a range of parameters for the case of houses and sylvatic populations.

To highlight the effect of spraying efficiency, arguably the most easily manipulated parameter, we show how R_0 varies as a function of spraying efficiency in houses and peridomestic habitats (corrals, Fig. 6). If <50% of vectors in corrals are eliminated by spraying, $R_0 > 1$, regardless of spraying efficacy in houses, and when spraying in corrals kills >90%, $R_0 < 1$.

Discussion

Because eliminating household vectors is the recommended method to reducing Chagas disease prevalence, we developed a household model to evaluate intervention strategies, such as spray rate and spraying efficiency, and optimize vector control. Our analysis suggests that the presence of a sylvatic or peridomestic cycle that is sprayed differently from houses and differs in insecticide persistence times leads to higher domestic infestation rates. The sensitivity of R_T to variations in the parameters depends on whether sylvatic vectors are present; specifically, the presence of sylvatic vectors can increase R_T by over an order of magnitude. When there are sylvatic vectors, spraying rate is relatively unimportant; com-

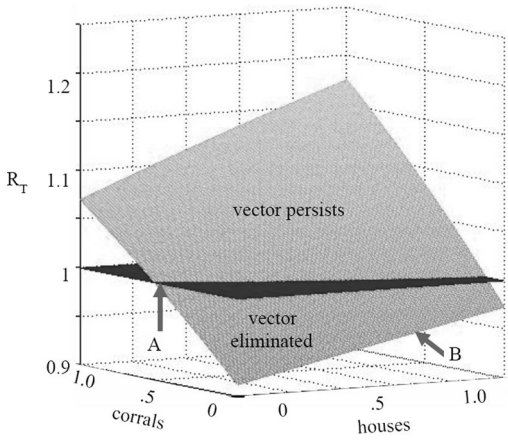

Fig. 6. Sensitivity analysis showing the effect of spraying efficiency in corrals and houses on reproduction number R_0. The dark blue plan is a reference for $R_0 = 1$. When $R_0 < 1$ (below the blue plane), vectors will be eliminated. Arrow *A* highlights that if <50% of vectors in corrals are eliminated by spraying, $R_0 > 1$, regardless of spraying efficiency in houses. Arrow *B* highlights the edge of the teal plane, showing that $R_0 < 1$ for when spraying in corrals kills >90% of vectors, regardless of efficacy in houses.

munity size, especially the ratio of houses to sylvatic burrows, is most important, followed by vector mobility. Infestation rate and spraying efficacy are relatively unimportant, and dwelling replacement rate has a large effect if houses persist for more than a few years.

The parameters vary in their facility of manipulation. Many, such as community size and house replacement rate, are intrinsic properties of the locality being examined. Others, such as vector movement between houses and between the two types of habitats, are influenced by a combination of human and extrinsic factors. A third group, most amenable to human management, includes spraying efficacy. Both ease of manipulation and effect on R_T need to be considered to help identify the most efficient and cost-effective approach to vector control. Further, some of these variables will change over time. For example, efficacy would be altered by the evolution of insecticide resistance or by the quantity of insecticide applied, as well as insecticide dissemination into refugia such as thatched roofs and cracks and crevices in the walls.

Recently, Lucero et al. (2013) reported that low-cost home improvements (i.e., construction of chicken coops away from houses and plastering of walls) that limit areas of vector refuge within the home are more effective at keeping infestation low and increase spraying efficacy because there are fewer places inside houses for vectors to hide. Our model supports these findings; Fig. 6 suggests that when the proportion of houses where vectors survive spraying is low, $R_0 < 1$, even if efficacy in corrals is 50%, but when there are many cracks and crevices within homes, vectors survive spraying and efficacy in corrals needs to be ≈90% for $R_0 < 1$.

Although developed in the context of Chagas disease, this model could be applied to other systems such as varroa mite infestation in honey bee colonies, a problem causing millions of dollars of damage each year. Mite infestation of hives follows a similar pattern to triatominae infestation of houses, and control efforts are similar, residual miticide treatments are applied at the level of the hive and on a periodic basis.

In conclusion, our model evaluates how local conditions affect the Chagas vector control strategy advocated by the World Health Organization. Further modeling attempts could include a more complex and stochastic approach. Although we consider both the ability to manipulate the parameters and their effect on R_T, our model does not include variation in space or time. In addition, model development could also consider the spatial structure using stochastic cellular automata. This model is a first step in developing data-based recommendations for control of Chagas disease vectors.

Acknowledgments

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References Cited

Anderson, R. M., and R. M. May. 1991. Infectious diseases of humans. Dynamics and control. Oxford University Press, Oxford, United Kingdom.

Andrade, J. P., J. A. Marin Neto, A. A. Paola, F. Vilas–Boas, G. M. Oliveira, F. Bacal, E. A. Bocchi, D. R. Almeida, A. A. Fragata Filho, C. Moreira Mda, et al. 2011. I Latin American Guidelines for the diagnosis and treatment of Chagas' heart disease: executive summary. Arq. Bras. Cardiol. 96: 434–442.

Buitrago, R., E. Waleckx, M. F. Bosseno, F. Zoveda, P. Vidaurre, R. Salas, E. Mamani, F. Noireau, and S. F. Breniere. 2010. First report of widespread wild populations of *Triatoma infestans* (Reduviidae, Triatominae) in the valleys of La Paz, Bolivia. Am. J. Trop. Med. Hyg. 82: 574–579.

Bustamante, D. M., M. C. Monroy, S. Pineda, A. Rodas, X. Castro, V. Ayala, J. Quinones, B. Moguel, and R. Trampe. 2009. Risk factors for intradomiciliary infestation by the Chagas disease vector *Triatoma dimidiata* in Jutiapa, Guatemala. Cad. Saúde Pública 25 (Suppl. 1): S83–S92.

Calderon, C. I., P. L. Dorn, S. Melgar, J. J. Chavez, A. Rodas, R. Rosales, and M. C. Monroy. 2004. A preliminary assessment of genetic differentiation of *Triatoma dimidiata* (Hemiptera: Reduviidae) in Guatemala by random amplification of polymorphic DNA-polymerase chain reaction. J. Med. Entomol. 41: 882–887.

Capinera, J. 2008. Encyclopedia of entomology, 2nd ed. Springer, New York, NY.

Cohen, J. E., and R. E. Gurtler. 2001. Modeling household transmission of American trypanosomiasis. Science 293: 694–698.

Cruz–Pacheco, G., L. Esteva, and C. Vargas. 2012. Control measures for Chagas disease. Math Biosci. 237: 40–60.

Dorn, P. L., S. Melgar, V. Rouzier, A. Gutierrez, C. Combe, R. Rosales, A. Rodas, S. Kott, D. Salvia, and M. C. Monroy. 2003. The Chagas vector, *Triatoma dimidiata* (Hemiptera: Reduviidae), is panmictic within and among adjacent villages in Guatemala. J. Med. Entomol. 40: 436–440.

Giordano, R., J. C. Cortez, S. Paulk, and L. Stevens. 2005. Genetic diversity of *Triatoma infestans* (Hemiptera: Reduviidae) in Chuquisaca, Bolivia based on the mitochondrial cytochrome b gene. Mem. Inst. Oswaldo Cruz 100: 753–760.

Hotez, P. J., M. E. Bottazzi, C. Franco–Paredes, S. K. Ault, and M. R. Periago. 2008. The neglected tropical diseases of Latin America and the Caribbean: a review of disease burden and distribution and a roadmap for control and elimination. PLoS Negl. Trop. Dis. 2: e300.

Inaba, H., and H. Sekine. 2004. A mathematical model for Chagas disease with infection-age-dependent infectivity. Math Biosci. 190: 39–69.

Lucero D. E., L. A. Morrissey, D. M. Rizzo, A. Rodas, R. Garnica, L. Stevens, D. M. Bustamante, and M. C. Monroy. 2013. Ecohealth interventions limit triatomine reinfestation following insecticide spraying in La Brea, Guatemala. Am. J. Trop. Med. Hyg. 88: 630–637.

Moncayo, A., and A. C. Silveira. 2009. Current epidemiological trends for Chagas disease in Latin America and future challenges in epidemiology, surveillance and health policy. Mem. Inst. Oswaldo Cruz 104 (Suppl. 1): 17–30.

Monroy, M. C., X. Castro, D. M. Bustamante, S. Pineda, A. Rodas, B. Moguel, V. Ayala, and J. Quinonez (eds.). 2011. An ecosystem approach for the prevention of Chagas disease in Rural Guatemala. Springer, New York, USA/ International develomment Research Centre, Ottawa, Canada.

Noireau, F. 2009. Wild *Triatoma infestans,* a potential threat that needs to be monitored. Mem. Inst. Oswaldo Cruz 104: 60–64.

Perez de Rosas, A. R., E. L. Segura, and B. A. Garcia. 2011. Molecular phylogeography of the Chagas' disease vector *Triatoma infestans* in Argentina. Heredity (Edinb) 107: 71–79.

Piccinali, R. V., P. L. Marcet, L. A. Ceballos, U. Kitron, R. E. Gurtler, and E. M. Dotson. 2011. Genetic variability, phylogenetic relationships and gene flow in *Triatoma infestans* dark morphs from the Argentinean Chaco. Infect. Genet. Evol. 11: 895–903.

Pizarro, J. C., and L. Stevens. 2008. A new method for forensic DNA analysis of the blood meal in Chagas disease vectors demonstrated using *Triatoma infestans* from Chuquisaca, Bolivia. PLoS One 3: e3585.

Pizarro, J. C., L. M. Gilligan, and L. Stevens. 2008. Microsatellites reveal a high population structure in *Triatoma infestans* from Chuquisaca, Bolivia. PLoS Negl. Trop. Dis. 2: e202.

Pizarro, J. C., D. Lucero, and L. Stevens. 2007. A method for the identification of guinea pig blood meal in the Chagas disease vector, *Triatoma infestans.* Kinetoplastid Biol. Dis. 6: 1–6.

Pizarro, J. C., W. Ribera, A. Aguirre, and A. De Muynck. 1996. Estudio parasitológico-entomológico de traiatomíneos procedentes de la provincia Zudáñez del Departamento de Chuquisaca. Arch. Boliv. Med. 3: 29–33.

Quisberth, S., E. Waleckx, M. Monje, B. Chang, F. Noireau, and S. F. Breniere. 2011. "Andean" and "non-Andean" ITS-2 and mtCytB haplotypes of *Triatoma infestans* are observed in the Gran Chaco (Bolivia): population genetics and the origin of reinfestation. Infect. Genet. Evol. 11: 1006–1014.

SEDES-Chiquisaca. 2006. Control vectorial del Servicio Departamental de Salud de Chuquisaca, Sucre, Bolivia.

Spagnuolo, A. M., M. Shillor, and G. A. Stryker. 2011. A model for Chagas disease with controlled spraying. J. Biol. Dyn. 5: 299–317.

Stevens, L., P. L. Dorn, J. O. Schmidt, J. H. Klotz, D. Lucero, and S. A. Klotz. 2011. Kissing Bugs: the vectors of Chagas. Adv. Parasitol. 75: 165–188.

Stevens, L., P. Dorn, J. Hobson, N. de la Rua, D. Lucero, J. Klotz, J. Schmidt, and S. A. Klotz. 2012. Evidence of human and other vertebrate bloodmeals and Trypanosoma cruzi infection in Chagas disease vectors from the United States. Emerg. Infect. Dis. 18: 646–649.

Tinsley, M., F. I. Lewis, and F. Brulisauer. 2012. Network modeling of BVD transmission. Vet. Res. 43: 1–9.

Velasco–Hernandez, J. X. 1994. A model for Chagas disease involving transmission by vectors and blood transfusion. Theor. Popul. Biol. 46: 1–31.

Waleckx, E., S. Depickere, R. Salas, C. Aliaga, M. Monje, H. Calle, R. Buitrago, F. Noireau, and S. F. Breniere. 2012. New discoveries of sylvatic *Triatoma infestans* (Hemiptera: Reduviidae) throughout the Bolivian Chaco. Am. J. Trop. Med. Hyg. 86: 455–458.

Waleckx, E., R. Salas, N. Huaman, R. Buitrago, M. F. Bosseno, C. Aliaga, C. Barnabe, R. Rodriguez, F. Zoveda, M. Monje, et al. 2011. New insights on the Chagas disease main vector *Triatoma infestans* (Reduviidae, Triatominae) brought by the genetic analysis of Bolivian sylvatic populations. Infect. Genet. Evol. 11: 1045–1057.

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